Human Health Risks

Assembled for Chapter 3

of the

Diamond Lake Restoration Final Environment Impact Statement

This compendium was assembled from the following specialist reports:

1) Human Risk Assessment for the Diamond Lake Restoration Project.
Barbara Fontaine, USFS and Angie Obery, Oregon DEQ. January 21, 2003. This 19 page report assessed the risks to human health of using rotenone under Alternatives 2 and 3.

2) Addendum to the Human Health Risk Assessment for the Diamond Lake Restoration Project—Alternative 5. Barbara Fontaine, August 20, 2004. This 9 page report assessed the risks to human health of using rotenone under Alternative 5 which was added to the FEIS as a result of public comment on the Diamond Lake DEIS.

3) Human Health Risk Assessment for the Diamond Lake Restoration Project—Toxic Algae Blooms. Jacob Kann, Ph.D. and Barbara Fontaine, USDA, February 10, 2004. This 12 page report addressed the risks to human health from toxic algae blooms under all alternatives 1, 2, 3, and 4.

In addition, this compendium represents disclosure on the effects to human health associated with the changes made to Alternative 4 between the Draft EIS and the Final EIS.

Barbara Fontaine Date:
Fisheries Biologist
Umpqua National Forest

HUMAN HEALTH RISK

Scoping identified a concern that the proposed rotenone treatment would present risks to human health and safety through exposure to rotenone. The potential effects to human health associated with the use of rotenone are tracked in this section. Additionally, because Alternative 1 proposes no active management at Diamond Lake, it is assumed that toxic algae blooms would continue to present human health risks. Therefore, the effects of toxic algae blooms on human health are also documented in this section.

TOXICITY OF ALGAE BLOOMS TO HUMANS

The blue-green algal blooms in Diamond Lake during the summers of 2001, 2002, and 2003 have presented risks to human health. Blue-green algae, also known as cyanobacteria, are single-celled aquatic plants found in surface waters worldwide that produce toxins. Such toxins have been implicated in human health problems ranging from skin irritation and gastrointestinal upset, to death from liver or respiratory failure (Chorus and Bartram 1999; Chorus 2001)

The two main species of toxin-producing blue-green algae associated with the blooms in Diamond Lake are *Anabaena flos-aquae* and *Microcystis aeruginosa*, with *Anabaena* more prominent (Eilers and Kann 2002). *Anabaena* is most frequently associated with the powerful neurotoxin, anatoxin-a; however, it can also produce the liver toxin, microcystins. *Microcystis aeruginosa* also produces microcystins, the liver toxin (Yoo et al. 1995). These species, like all blue-green algae, have compounds in their cell walls that are responsible for the adverse skin, eye, mucosal, and digestive reactions reported by people who have come in contact with them (Chorus 2001).

Anatoxin-a[1]

Anatoxin-a affects the nervous system, often leading to convulsions and death by suffocation (Carmichael 1994). Animals exposed to anatoxin-a through drinking *Anabaena*-contaminated water, grooming scum from their coats or feathers, or in laboratory tests, die of the poison within minutes or hours. Cattle, pet dogs, waterfowl and others have met their deaths in this way (Backer 2002).

In 2002 the first human presumed to die from *Anabaena* toxin in the United States occurred near Madison, Wisconsin, when a teenage boy died 48 hours after diving and swimming in a scum-coated pond. Several others who went into the pond at the same time also developed gastrointestinal symptoms, but only one, who had also gone completely underwater, became acutely ill with diarrhea. However, because of the length of time between exposure and death, some uncertainty remains as the whether algal toxins were the direct cause of death (Milwaukee Journal Sentinel, 2002).

Insufficient data exists to establish reliable safety thresholds for human exposure to anatoxin-a. Human studies are lacking and although there is experimental laboratory data generated

[1]Anatoxin-a is an alkaloid that acts as a post-synaptic depolarizing neuromuscular blocking agent, causing nerve impulses to over-stimulate muscle cells, particularly those involved in breathing.

from mice, it is considered inadequate for the formulation of human Tolerable Daily Intake Standards, whether for toddlers or adults (Chorus and Bartram 1999). However, Dr. Wayne Carmichael of Wright State University suggested that 100 micrograms per liter of water would present a lethal risk to pets (Table 40) drinking from shoreline areas where blooms tend to be concentrated. In 2001, anatoxin-a concentrations were detected as high as 300 ug/L (micrograms per liter of water) in Diamond Lake.

Uncertainty exists as to accumulation of anatoxin-a in fish tissue; fishermen may be at risk in eating their catches (Falconer 1993). Finally, although it does not appear to be the case that recurrent low exposure to anatoxin-a leads to health problems later on, this is not established, and there is concern that people repeatedly exposed may become sensitized and develop increasingly more severe reactions with each new exposure (Backer 2002).

Microcystins[2]

Microcystins were first isolated from *Microcystis aeruginosa*, but are also produced by other species, including *Anabaena*. These powerful liver toxins disrupt the structure of liver cells, causing cell destruction, liver hemorrhage, liver necrosis, and death (Carmichael 1994).

Microcystins in the water supply of a renal (kidney) dialysis clinic in Brazil resulted in the illness of 110 of 113 patients, including the deaths of 55 patients from liver hemorrhage and liver failure (Backer 2002). Microcystin delivered by the nasal route to experimental rodents also resulted in erosion of the nasal mucosa to the point of hemorrhage. In addition to liver toxicity, long-term laboratory animal studies indicate that microcystins promote tumors and birth defects (Falconer et al. 1988). Anecdotal evidence for such chronic effects on humans is based in large part on the high rates of liver cancer in the rural regions of China where drinking water was obtained from ditches and ponds with large blue-green algae loads. Where drinking water supplies have been changed to deep wells, cancer rates have begun to drop.

Microcystin poisoning has been implicated in the largest number of blue-green algae-associated animal deaths worldwide, and enough work has been done, both with rodents and pigs on microcystin effects at various levels of exposure, that the World Health Organization (WHO) has issued a provisional guideline of 1 microgram per liter of water (ug/L) for microcystin concentration in drinking water. Microcystin concentrations found during three summers sampling and testing the waters of Diamond Lake have been less than 1 ug/L except one sample in 2003, which was 2.54 ug/L, exceeding the preliminary WHO guideline.

The exposure risks associated with microcystin toxins are exacerbated over that of Anatoxin-a, because the toxins continue to release into the water, remaining even after the *Microcystis* bloom has visually dissipated (Lam et al. 1995). Thus, microcystin has the potential of exerting continued risk to human health when it may not be obvious.

[2]Microcystins are hepatotoxins (liver toxins) which are powerful cyclical peptides which disrupt the structure of liver cells.

Table 40. Summary of the toxicity information available on algae toxins.

	Anatoxin-a	Microcystin
Proposed Safe Concentrations	Not established—100 ug/l would likely kill a dog drinking from the lake (Carmichael),	1 ug/kg/day (Proposed by World Health Organization)
Levels observed in D. Lake	Up to 300 ug/L	Up to 2.54 ug/L
Non-acute effects	unknown	Probably carcinogenic

TOXICITY OF ROTENONE TO HUMANS

Under Alternatives 2 and 3, the liquid formulation of rotenone, Noxfish®, would be applied to Silent and Short Creeks only, while the powdered formulation of rotenone, Pro Noxfish®, would be applied throughout the areas of the lake. Thus, Alternatives 2 and 3 are identical with respect to the application of rotenone. Alternative 5 was developed as a result of public comments received during the DEIS comment period. It was suggested by expert personnel of the California Department of Fish and Game, that to achieve more certainty for full tui chub eradication, liquid rotenone should be used in the shallow areas of the lake occupied by aquatic plants. The liquid formulation is more effective in such environments, because it disperses more quickly and thoroughly than the powder and has a higher likelihood of killing all the fish present at the time of application. Rotenone products have been classified by the US Environmental Protection Agency (USEPA) as Category 1 materials which are in the "extremely toxic" range for acute (short-term) toxicity. Laboratory mammals are used to assess the levels of toxicity. The Extension Toxicology Network[3] affiliated with several prominent Universities across the United States summarized the following information from the scientific literature on rotenone toxicity in mammals (ExToxNet, 1996). In acute oral exposure studies, where large doses are fed to test animals over a short time, rotenone was found to be slightly to moderately toxic to mammals. Reported oral LD50 values range from 132 to 1500 mg/kg of body weight in rats. The LD50 is the amount of ingested material that would be lethal to the average laboratory mammal. When 50 percent of the animals in the experiment die, the average lethal dose (LD) is established. Ingested rotenone is believed to be moderately toxic to humans with an oral lethal dose estimated to be between 300 to 500 mg/kg of body weight. Human fatalities are rare perhaps because rotenone is not widely used and because its irritating action causes vomiting (ExToxNet, 1996). Both the liquid and powered formulations of rotenone in their undiluted states are reported to be potentially fatal to humans if inhaled or ingested. Ingestion or inhalation can cause numbness, nausea, vomiting, and tremors.

The rotenone formulations are moderately to highly toxic when inhaled and are therefore considered more toxic when inhaled than when ingested. In rats and dogs exposed to

[3] ExToxNet is a pesticide Information project of cooperative Offices of Cornell University, Oregon State University, the University of Idaho, University of California at Davis, and the Institute for Environmental Toxicology at Michigan State University.

rotenone dust, the inhalation fatal dose was uniformly smaller than the oral fatal dose. Fifty percent of female rats died when exposed to a concentration of 0.045 mg/liter of air over a 4 hour period (Prentiss Incorporated, 2000b). A spray of 5% rotenone in water was fatal to a 100-pound pig when exposed to 250 mL of the airborne mixture (ExToxNet, 1996).

In chronic toxicity studies, where non-lethal doses are fed to laboratory animals over extended periods of time, rotenone has been found to have low levels of toxicity when ingested. Dogs fed rotenone for 6 months at doses up to 10 mg/kg/day showed reduced food consumption and therefore weight loss. At the highest doses, blood chemistry was adversely affected possibly due to gastrointestinal lesions and chronic bleeding (ExToxNet, 1996).

The rotenone formulations proposed for use in Alternatives 2, 3, and 5 are reported to be slightly toxic to non-irritating to the skin from dermal exposure. Dermal exposure to rotenone can cause skin and eye irritation. The lethal dose to rabbits from skin absorption of the powdered formulation was greater than 2,020 mg/kg (Prentiss Incorporated, 2000a).

Other toxic effects of rotenone have also been characterized by studies with lab animals and summarized by ExToxNet (1996) and USEPA Integrated Risk Information System (IRIS) (2003). Reproductive toxicity was established in a two generation rat reproduction study conducted by the USEPA in 1983 (IRIS, 2003). Rats fed at 1.88 mg/kg/day (equal to 37.5 ppm) exhibited the lowest effect level for reproductive toxicity while rats fed 0.38 mg/kg/day (equal to 7.5 ppm) exhibited the no-observed effects level. Whether rotenone is teratogenic (causes birth defects) is not known since a feeding study of pregnant rats showed skeletal deformations in rat pups at low doses, but no deformities at higher doses. Rotenone was found not to be mutagenic (cause changes in the genetic material of cells) in treated mice and rats based on several studies at the cellular level. Most rodent studies have revealed no evidence of carcinogenic activity and the prevailing scientific opinion is that rotenone is not carcinogenic (USEPA, 1981 and 1989). USEPA last conducted a comprehensive review of rotenone in 1988 and re-registration is tentatively scheduled for 2006.

A recent study (Betarbet et al, 2000) reported that rats injected with rotenone at 2 to 3 mg/kg body weight each day in the jugular vein for 5 weeks showed symptoms similar to that of Parkinson's disease. Other chemicals were administered with the rotenone to enhance tissue penetration. None of the other studies that used realistic exposure pathways (oral, inhalation or dermal) of rotenone have reported such findings. Betarbet et al (2000) demonstrated that rotenone is an inhibitor of one of the five enzyme complexes in cells of test mammals. Neurological research continues to explore the link between Parkinson's disease and pesticide exposures (such as rotenone and others). Although the exact cause of Parkinson's disease is unknown, recent epidemiological studies suggest an association with single gene mutations, toxic exposures, or some combination of the two factors (Greenamyre et al, 2003). The USEPA has reviewed this study and is determining the appropriate course of action. The results of this review will help determine what next steps the USEPA will take towards the completion of the next rotenone review (USEPA 2003).

Inert Ingredients, Metabolites, and other Chemicals used with Rotenone

Chemical manufacturers often add other ingredients to their formulations, called inert ingredients, to enhance effectiveness. The powered formulation, Pro Noxfish® that would be applied to Diamond Lake has no added inert ingredients; it is composed simply of the ground up plant material. The liquid Noxfish® that would be applied to Short and Silent Creeks, as well as the shallow portions of the lake under Alternative 5 contains inert emulsifiers, solvents, and carriers that are important in ensuring the solubility and dispersion of this liquid formulation. Water treated with Noxfish® was found to contain rotenolone (the metabolite of rotenone), and volatile organic compounds (trichloroethylene, xylene, toluene, and trimethylbenzene) and semi-volatile organic compounds (naphthalene, 1-methyl naphthalene, and 2-methyl naphthalene). These volatile and semi-volatile organic compounds naturally breakdown and dissipate in treated water before rotenone and rotenolone (Finlayson et al. 2000).

Five California rotenone projects were monitored for the fate of the compounds of powdered and liquid formulations including inerts in sediments (Finlayson et al, 2001). Only the naphthalene and methyl naphthalene (associated with Noxfish®) temporarily accumulated in sediments, but this was for a period of less than 8 weeks. The other inert compounds in Noxfish® did not persist in sediments.

Nine California rotenone projects were monitored for the inert ingredients in Noxfish® in surface water (Finlayson et al, 2001). All ingredients were well below the minimum concentrations allowed under maximum contaminant levels (MCLs) for these ingredients in drinking water standards set by the EPA (Finlayson, 2001). Of the seven organic compounds found in Noxfish, trichloroethylene (TCE) is the only carcinogen; the rest are considered noncarcinogens. However, there are inconsistencies in the scientific literature regarding whether naphthalene is carcinogenic. Naphthalene was reported in one source as causing carcinogenic activity in rat nose tissue in an inhalation study (US National Toxicology Program, 2001). The bulk of the toxicology literature however, supports that naphthalene is not carcinogenic.

Following application of Noxfish®, samples collected during application into flowing water did not detect TCE (<0.5 ug/L) or xylene (<0.5 ug/L) except for one sample collected immediately below a drip station at 0.76 ug/L TCE and 0.56 ug/L xylene. Naphthalene and 2-methylnapthalene were detected at concentrations ranges of <0.5 to 57 ug/L and <2 to 50 ug/L.

The USEPA has established drinking water standards for levels of rotenone and associated chemicals (Table 41).

Table 41. Human Health Standards, Risk-based Safe Levels, and Detection Limits for Rotenone and Other Associated Ingredients in Drinking Water.

Fish Toxicant Ingredients	Maximum Contaminant Level[1] (ug/L)	Maximum Contaminant Level Goal[1] (ug/L)	Preliminary Remediation Goal[2] (ug/L)	Analytical Detection Limit (ug/L)	Analytical Method
Rotenone	Not Available	Not Available	150	50	SDWA EPA Method 553 (HPLC)
Naphthalene	Not Available	Not Available	6.2	0.5	SWDA EPA Method 524.5
Toluene	1,000	1,000	720	0.5	SWDA EPA Method 524.5
Trichloroethy-lene	5	Zero	0.028	0.006[3]	USEPA 8260 Mod SIM
Trimethylben-zene	Not Available	Not Available	Not Available	0.5	SWDA EPA Method 524.5
Xylene	10,000	10,000	210	0.1	USEPA 8260 Mod SIM

NOTES:
1. USEPA 2002b Based on safe drinking water standards.
2. USEPA 2002a Based on safe risk-based levels for residential tap water use
3. Value provided is the MDL instead of the reporting limit. The reporting limit for TCE is 0.05 ug/L using EPA Method 8260 Mod GCMS-SIM.

MCL – maximum contaminate level. The highest level of a chemical allowed in drinking water. It is an enforceable level under the Safe Drinking Water Act.
PRG - preliminary remedial goal. The level of a chemical in drinking water that is not expected to cause any adverse effects for a lifetime of exposure. Lifetime exposure is based on 30 years of exposure for a child and adult drinking 1 and 2 liters, respectively.
Analytical Detection Limit. The level at which a chemical can be accurately and precisely quantified by a certain method.
SWDA – Safe Drinking Water Act. Gives EPA the authority to set drinking water standards. Used in the context of analytical methods developed under the SWDA program for monitoring water quality.
RCRA - Resource Conservation and Recovery Act. Used in the context of analytical methods developed under the RCRA program for monitoring water quality.

The possible metabolites of rotenone are carbon dioxide and a more water soluble compound (rotenolone) that is excreted in the urine. Studies indicate that approximately 20 percent of applied oral doses are eliminated from the animals system within 24 hours.

Potassium permanganate is a neutralizing agent that would be transported with rotenone and stored on site as a precautionary measure. It has no deleterious effects at the concentrations normally associated with the neutralizing process (Finlayson et al, 2000). However, in its concentrated form, it is caustic to mucous membranes in the nose and throat. The required protective clothing and breathing apparatus when handling the concentrated powder would lessen human health risks. This neutralizing agent is commonly transported and stored with rotenone as a precautionary measure in case rotenone is spilled or otherwise escapes into non-target water bodies. Use of potassium permanganate is not proposed under this alternative and it is not considered reasonable to assume that it would be used in association with the project. During the time period when rotenone would be stored at Diamond Lake, headgates would be closed on the canal and Lake Creek, and the first 5.5 miles of Lake Creek would be dry. Rotenone treated waters would be confined within Diamond Lake. There are no foreseeable situations that would warrant neutralizing a spill in the project area because the available waters in proximity to storage sites would be scheduled for rotenone treatment and thus, a spill would not present a problem requiring neutralizer.

AFFECTED ENVIRONMENT

TOXIC ALGAL BLOOMS

Extensive amounts of blue-green algae in Diamond Lake over the last few years produced toxins to the point of risking human health. The large population of tui chub in Diamond Lake has the potential to contribute to the hazardous algal blooms in two ways. First, tui chub can eat the larger-sized zooplankton reducing their populations to the point that they no longer effectively graze on the algal cells and thus no longer keep the algal population in check. Secondly, the tui chub population can increase the nutrient concentration in the lake through excretion of nitrogen and phosphorus in forms available for algal growth, essentially adding fertilizer for the plant population to expand. These ecological changes in conjunction with optimal climatic conditions of warm, sunny weather and calm, fertile water may have enhanced the blooms in Diamond Lake.

Alert level guidelines based on cell counts have been established for blue-green algae blooms in drinking and recreational waters (Yoo et al. 1995; Chorus and Bartram 1999). Alert Level 1 is 500 cells/ml and requires increased frequency of monitoring; Alert Level 2 is 2000 cells/ml and requires alerting the public and posting water bodies; Alert Level 3 is 15,000 cells/ml and warrants a recreational closure for water contact recreation. Alert Level 3 was shown in a study to result in skin and mucosal irritation and gastrointestinal symptoms. Diamond Lake has reached Alert Level 3 during the summer months of 2001-2003. Consequently, Diamond Lake was closed to swimming and all water contact from August 10 to August 30, 2001; July 23 to August 7, 2002; and from July 1 to August 12, 2003—a total of 46 days over three summers. Due to diligent monitoring and lake closures, no people are known to have suffered illness from exposure to blue-green algae toxins thus far at Diamond Lake.

The amount of toxin produced by the blue-green algae in Diamond Lake is highly variable, but it is generally related to their density or sheer amount in the water column (Chorus and Bartram 1999). Moreover, because these blue-green algae cells are buoyant and frequently

concentrate at the surface, wind may concentrate cells along the shoreline or in bays, causing toxins to be at sufficiently high levels, and sufficiently accessible, to be dangerous to humans who come in contact with them (Chorus and Bartram 1999).

The blooms make the affected water disagreeable such that people tend to voluntarily limit their exposure. Although few people would be likely to actively drink from such a source, the possibility of recreational exposure through accidental immersion and ingestion or aspiration exists, especially if boating activities are going on. Even a scummy lake can look irresistible on a blazing afternoon, and if mats of bloom wash up onto the shoreline, there may be considerable risk to young children (Chorus 2001).

It is also difficult to know how much toxin is actually in Diamond Lake at any given time. Tests to determine toxin levels take time; the bloom may be in a period of exponential growth, with toxin levels changing fast, or be dying off, in which case breakdown of the cells may be releasing large amounts of toxin into water that looks free of problems. Wind and wave motion may concentrate the bloom and its toxins in one part of the lake, leaving other areas apparently free of problems.

Under current conditions of extremely high numbers of tui chub, toxic blue-green algae blooms (and subsequent lake closures) will likely continue, with the severity determined by the variability in climate during the summer. Under current high nutrient excretion rates by tui chub, the main determinant of yearly toxic bloom variability will depend on the occurrence of the calm, sunny, warm conditions that tend to favor blooms of blue-green algae (Kann 2003). However, under a given set of climatic conditions the likelihood of large algae blooms are predicted to be diminished (although periodic blooms can still be expected) as available nutrients decrease in the water column if tui chub were removed (Kann 2003).

Though surface waters are at continued risk of contamination, groundwater is not susceptible to contamination from toxic algae blooms. Chorus and Bartram (1999) assessed groundwater for the presence of algal cells and algal toxin. They found extreme low levels of both algae cells and algae toxins in ground water. These authors reported that from 93.7 to 99.7 percent of the algae cells and 97.5 to 99.5 percent of the toxins were filtered out of the surface water as it traveled through the sediments and rock substrate of the ground water environment.

ENVIRONMENTAL EFFECTS

TOXIC ALGAL BLOOMS

Direct Effects:

Direct effects to human health would be those that occur in the short-term, over a period of several years, and at the immediate site of Diamond Lake itself.

Under Alternative 1, the general visiting public would continue to be at risk of exposure to lake waters contaminated with blue-green algae toxins. Elevated concentrations of either anatoxin-a or microcystins, or both, can be expected to continue during warm, sunny weather

as long as tui chub remain abundant in Diamond Lake. The likelihood of exposure would be lessened by annual water monitoring for the algae toxins that would be used to alert lake users and to trigger necessary lake closures.

However, as time goes on, the chances of public exposure by accident or by ignoring the warnings and closures would increase simply due to the long-term presence of the risk coupled with the popularity of Diamond Lake to the recreating public. Since the concentrations of toxins can be variable in the lake depending on location, depth, shoreline configurations, wind effects and rapid algal population shifts, the toxicity monitoring may not catch hot spots of concentrated toxins and timely lake closures may not always occur. In this event, swimmers or boaters could potentially receive a dose of the toxin(s) and become seriously ill. If such a dermally exposed person were to accidentally swallow concentrated toxins at the same time, there may be a potential of death, such as may have occurred in Wisconsin in 2002. However, the probability of mortality is still extremely low.

Agency employees or private contractors involved in the toxicity monitoring would also be at continued risk under Alternative 1. Since these administrative workers would be seeking out areas of concentrated toxins, the consequences of accidental submersion would be relatively high. Such an accident could result in the direct effect of serious illness or even death if some of the toxin is swallowed during an accident. These potential direct effects to the health of administrative personnel would be lessened by safety training and the implementation of pre-planned mitigation such as washing with clean water immediately following an incident.

Under Alternative 1, the domestic water from both the shallow and deep aquifer wells would not be at risk of contamination from algal toxins. This is because the toxins are expected to be filtered out of the water by the sediments and rock that exists in the ground water environment (Chorus and Bartram, 1999).

Under Alternatives 2, 3, and 5, prior to the rotenone application when the lake would be drawn down by about 8 feet in elevation, toxic algae blooms could be significant if weather conditions were conducive to blooms. Moreover, wave action on exposed sediments along the dewatered shoreline coupled with boat-generated turbulence could release more phosphorous than would normally occur if the lake was full of water. Under the right weather conditions, the disturbed sediments associated with implementation of Alternatives 2, 3 or 5 could lead to more algae growth and potentially more risk to the workers involved in implementing any of these alternatives. The potential of exacerbated algae growth, over and above that predicted under Alternative 1, is expected to be limited however because the phosphorus available for plant growth (i.e. dissolved in the water column) has a strong tendency to bind to sediment particles. Its residence time as dissolved phosphorus in the water column would depend on the amount of oxygen available in the lake water at the time. However, rapid uptake by sediment particles is expected (Johnson, 2003) with only a limited possibility of enhanced algae population growth under Alternatives 2, 3, and 5.

Because scores of people would be involved in the implementation of Alternatives 2, 3, and 5, the potential of administrative or application worker exposure to algae toxins is higher than the risk to administrative personnel only charged with water monitoring under Alternative 1.

This is simply because more people would be at risk of accidental exposure to algae toxins under Alternatives 2, 3, and 5, compared to Alternative 1 over the short-term.

Alternative 4 is expected to result in direct short-term public health hazards associated with exposure to toxic algae similar to that disclosed under Alternative 1. This is because Alternative 4 is expected to take about 7 years to meaningfully affect tui chub populations, the primary driver of the toxic algae blooms. During this time frame, the people who visit and live at Diamond Lake would be susceptible to all the exposure pathways to anatoxin-a and microcystins described under Alternative 1. Alternative 4 presents the most risk to the health of administrative personnel from blue-green algae toxins because of the intense labor involved in fish removal and the sheer number of workers that would have to venture out on the lake to carry out the adaptive fish removal process every September, when a risk exists of exposure to algae toxins.

Indirect Effects:

Indirect effects to human health are those that would occur downstream of Diamond Lake and effects that would occur in the future, over the long-term.

The consumption of water or dermal exposure of water downstream of Diamond Lake in Lake Creek could potentially put members of the public at risk of illness under all alternatives. This downstream, indirect effect is only possible during the same risk periods of high algal toxin concentrations experienced in Diamond Lake itself. The blue-green algae toxins in Lake Creek would only be those delivered from Diamond Lake. Since Lake Creek has turbulent flow that is not conducive to algae proliferation, no additional growth of algae or associated toxic releases are expected to originate in Lake Creek itself. The likelihood of anyone receiving a dose of blue-green algae toxins out of Lake Creek would be lessened by the heightened public awareness and lake closures that would be put into effect as necessary, under all alternatives. These potential downstream indirect effects would be sustained over long periods of time under Alternatives 1 and 4, but would be short-lived under Alternatives 2, 3, and 5, since Alternatives 2, 3, and 5 all rapidly reduce the tui chub population. However, an exact time period of the response of algal blooms to tui chub removal can not be determined.

The risk of any indirect effect from toxic algae to the health of Lake Creek users would progressively lessen in a downstream direction as tributary streams, with no connection to Diamond Lake, enter Lake Creek and dilute toxin concentrations. Likewise, the risks to human health from exposure to algae toxins in Lemolo Lake, located 12 miles downstream of Diamond Lake, is much less than that of Lake Creek. No indirect downstream effects to human health at Lemolo Lake are expected to occur under any of the alternatives. This large reservoir is fed by many tributary streams, including the North Umpqua River, that function to mix and dilute blue-green algae toxins below levels of concern (Hofford 2003). No testing for blue-green algae toxin has occurred in Lemolo Lake.

Under Alternative 1, the indirect effects to human health over long periods of time are expected to be similar to the direct effects to human health disclosed above; the potential of serious illness or even death from toxic algae would be present through the same exposure pathways. However, the chances of actually experiencing impacts to human heath associated

with Alternative 1 increase with time simply due to the additive effects of summer after summer of potential public and administrative worker exposure. As time passes, assuming a fairly static population of tui chub, weather conditions could align to create toxic concentrations above those experienced to date. On the other hand, cooler more overcast summers would result is fewer blooms and lower toxic concentrations. The threat of realizing indirect effects to human health over the long-term, under Alternative 1, can be expected to gradually increase over the years if the regional weather warming patterns continue on the same trajectory as the last few decades.

No long-term impacts to human health from exposure to toxic algae booms are expected under Alternatives 2, 3, or 5. Instead, Alternatives 2, 3, and 5 are expected to result in indirect beneficial effects to human health hazard levels in the long-run relative to the existing condition. Predicting the effect of tui chub removal on the magnitude and annual trends of toxic algal blooms in Diamond Lake is difficult due to uncertainty in such factors as inter-annual climatic variability, restocking of the lake with rainbow trout, internal nutrient recycling from the sediments, and response of the zooplankton and benthic communities. Nonetheless, it is clear based upon paleolimnological data, that the lake began changing shortly after being stocked with trout, and that the greatest increases in *Anabaena* were associated with increased populations of tui chub (Eilers et al, 2001). Alternatives 2, 3, and 5 are therefore expected to result in a long-term lessening of risks to human health relative to Alternative 1. If some tui chub survive the rotenone treatments under Alternatives 2, 3, and 5, or if they are reintroduced and contingency plans fail, negative impacts to the water quality and human health similar to the current condition could potentially occur again. However, the contingency plan included in these alternatives may help to slow or alleviate future water quality problems if tui chub do recur.

Under Alternative 4, the indirect effects to human health over long periods of time are expected to be similar to the indirect effects to human health disclosed above for Alternative 1; the potential of serious illness or even death from toxic algae would be present through the same exposure pathways. However, under Alternative 4, the gradual long-term decline of tui chub can be expected to result in fewer toxic algae blooms, lessening public health risks proportionately. Table 42 summarizes the risks to the various groups of people if exposed to toxic algae in Diamond Lake.

Table 42. Summary of Exposure Risks to the Algal Toxins Associated with the Diamond Lake Restoration Project.

Alternative	Administrative or Application Workers	General visiting public and other residents
1	4 workers every summer at high risk of accidental exposure to algae toxins (during water sampling) over multiple summers for the foreseeable future.	Hundreds of water recreationists (swimmers, boaters, water skiers, sailboarders) could be exposed to algae toxins every summer if monitoring does not adequately detect toxin levels and lake closures are not timely.
2, 3, & 5	25 workers at high risk of accidental exposure to algae toxins (during applications of rotenone from boats) and fish toxicants (while handling the concentrated formulations) during 1 summer. Potential exposure of 1-2 workers during water monitoring for the first few years of the project.	No long-term exposure anticipated unless chub remain after rotenone treatment or are introduced such that their populations eventually explode again. The contingency plan may help to slow or alleviate future risks to human health if tui chub do recur.
4	15 workers/summer at high risk of accidental exposure to algae toxins during both water monitoring and fish removal work over multiple summers for the foreseeable future.	Initially, hundreds of water recreationists (swimmers, boaters, water skiers, sailboarders) could be exposed to algae toxins every summer if monitoring does not adequately detect toxin levels and lake closures are not timely. The risk would incrementally decline with time as chub numbers decline with mechanical removal.

AFFECTED ENVIRONMENT

ROTENONE

There are only two possible pathways of public exposure to the rotenone formulations proposed under Alternatives 2, 3, and 5 of this project--either eating contaminated fish or drinking contaminated water. The other possible exposure pathways, dermal exposure and inhalation exposure, would not be possible for members of the public. No dermal exposure associated with the public swimming or wading in the treated waters is expected because the rotenone would not be concentrated enough once it has been mixed in the lake to lead to any concerns regarding dermal exposure (Finlayson et al. 2000). Rotenone product labels state that swimming would be allowed once the product has been mixed into the water (Prentiss 2000). Moreover, no member of the public would have access to the concentrated formulations to receive a meaningful dermal dose. Similarly, no public exposure via inhalation of either rotenone formulation is expected since the work areas where such dose pathways are possible would be under tight security with no public entry allowed. Airborne drift into adjacent area was found to be 1000 times less toxic than the no observed effect level of the chemical (Finlayson et al. 2000).

It would be extremely unlikely that members of the public would have access to dead or dying fish in order to unwittingly consume any contaminated fish and receive a dose of the fish toxicant. This is because Diamond Lake would be closed to public entry during the treatment period; public awareness of the closure would be heightened well in advance of the treatment; and because warning signs would be posted throughout the area. Since the fish in

Diamond Lake are expected to be rapidly killed by the treatment within a 2 to 3 week period, there would be no lingering danger of anglers ignoring the warnings and potentially angling and consuming fish. The large number of carcasses would be further disincentive for human consumption. No fish would be restocked into Diamond Lake until well after all residues are gone.

The primary pathway for members of the public to be exposed to rotenone is by drinking well water. This pathway presents more risk than the consumption of tainted fish because the water would essentially look and smell normal a few days following the application. The water in the lake itself presents the greatest risk to potential water drinkers, while waters downstream of the lake present little to no risk of public exposure. The risk of consuming contaminated water would be prevented by supplying drinking water to well users if rotenone or other added ingredients are detected in any of the monitoring wells. Based on the groundwater transport and modeling assessment, the Forest Service monitoring wells are adequate to intercept any rotenone or other added ingredients far in advance of transfer to a domestic well.

The consumption of contaminated surface water out of Diamond Lake by visiting members of the public is unlikely given the closure to public entry, the heightened public awareness, and the warning signs that would be in place throughout the area. These mitigation measures would remain in force until all risk of exposure is eliminated.

The potential for contamination of the groundwater is lessened somewhat however, due to the strong tendency of rotenone to attach to soil particles and organic sediment such as that found in the lake bottom. The lake sediments are expected to rapidly capture and hold the chemical, essentially "filtering" it out of the water column as the water from the lake enters the groundwater environment.

The primary concern for public consumption of tainted drinking water is associated with the domestic water users of the Diamond Lake area—primarily the users of shallow wells that service the summer homes on the west shore of the lake (Figure 4). A study conducted by the Forest Service of the potential for groundwater contamination was done for this project (Breeden, 2003). A total of 16 monitoring wells were installed to investigate groundwater movements around the lake. The study showed that the shallow aquifer on the north and northwest shores of Diamond Lake, which supplies most of the domestic wells in that area, can be expected to receive groundwater originating from the Lake during the fall and winter months. If treated water from the lake does in fact enter the shallow aquifer that supplies the west shore domestic users, then health risk could potentially exist for as long as 8 weeks or until the toxicant fully breaks down.

There are 102 summer homes located on west shore of Diamond Lake and all but about 20 are serviced by wells tapped into the shallow aquifer that is susceptible to contamination from treated lake water. Those summer home wells not at risk are the deeper wells, typically greater than 60 to 100 feet deep that tap into the deep aquifer. The Oregon Department of Fish and Wildlife maintains a small work facility at the Lake Creek outlet. The drinking water for this facility comes from Lake Creek which would be dry during the period of concern, so no exposure from the drinking water at this location is possible.

The draw down of the lake would result in no surface water flowing out of the lake at its only natural outflow, Lake Creek, or at the constructed drainage canal plumbed into Lake Creek. The draw down would occur during the winter months prior to the proposed September treatment. By September of the year of treatment, during the lowest flows of the year, the lake level is expected to be about 8 feet in elevation below the Lake Creek outlet. The chance of a fall rainstorm large enough to rewater the lake causing contaminated water to flow out of the Lake is extremely low. More than 20-inches of rain would need to fall to rewater the lake to the point of spilling over into Lake Creek during the two month period between around September 15 to November 15. Based on historic weather data, the average rain fall for this time period was 7.5-inches and the probability of receiving as much as 20 inches is extremely low. However, a mitigation measure is incorporated into Alternatives 2, 3, and 5 (Chapter 2) that would reconstruct the outflow structure of Diamond Lake in Lake Creek to an elevation that would contain any unexpectedly large amount of rainfall during this two month period.

Alternatives 2, 3, and 5 include a mitigation measure requiring the closure of these two outlets with headgate structures until tests indicated that rotenone, rotenolone[4], and all semi-volatile and volatile organic compounds[5] associated with the chemical treatment had dissipated to non-detectable or trace levels in both the water column and lake bottom sediments (approximately one to two months). The rotenone and its byproducts including the inert ingredients found in the liquid formulation, would be fully broken down prior to any downstream delivery of surface waters and associated sediments. Therefore, no downstream public exposure associated with surface water consumption is expected in Lake Creek or Lemolo reservoir, 12 miles downstream of Diamond Lake.

The Forest Service also investigated the potential of contaminated groundwater to discharge into Lake Creek. In September 2003, the Forest Service conducted a groundwater seepage study along a six mile length of Lake Creek. The results of this study showed that Lake Creek received no appreciable increase in flow due to groundwater discharge into the creek. No contamination of Lake Creek and its downstream areas, including Lemolo reservoir from the groundwater aquifer is expected. Groundwater discharge at a location further downstream of the six mile study area was not examined. However, the longer travel time to any potential discharge locations further downstream, the tendency for rotenone to bind with soil particles at the bottom of the lake, and the tendency for the rotenone to breakdown over time, all make the potential of groundwater contamination of downstream water bodies such as lower Lake Creek and Lemolo reservoir very remote.

The required mitigation measure under Alternatives 2, 3, and 5, of monitoring the waters of Diamond Lake and Lake Creek following the rotenone treatment will confirm whether or not any downstream waters contain any trace of the toxicant. If any residues are detected, the

[4] Rotenolone is the metabolite (by product) of rotenone (Finlayson et al. 2000).

[5] The liquid rotenone formulation Noxfish® contains inert emulsifiers, solvents, and carriers that are important in ensuring the solubility and dispersion of rotenone in water. Waters treated with Noxfish® may contain rotenone, rotenolone, and volatile (xylene, trichloroethylene, toluene, and trimethylbenzene) and semi-volatile (naphthalene, 1-methyl naphthalene, and 2-methyl naphthalene) organic compounds. These volatile and semi-volatile organic compounds dissipate in treated water before rotenone and rotenolone (Finlayson et al. 2000).

exposure to members of the public through drinking water from wells around the reservoir would be minimized by public notification, warning signs, supplied bottled water, and/or potential closures that would be put into effect. In the very remote case that the water supplies from Lake Creek and/or Lemolo are threatened, actions to minimize exposure would be taken.

The most likely individuals to be exposed to rotenone formulations proposed for this project are the application workers who will be involved in removing the concentrated formulations from their original containers, diluting, and mixing the formulations, filling application containers, and applying the rotenone out of boats in the lake and at drip stations in Silent and Short Creeks. At each step, the risk of accidental exposure is present. The primary exposure pathway would be via inhalation of the powdered formulation, Pro-Noxfish®, when rotenone become airborne once removed from its container and handled. The primary exposure pathway for the liquid Noxfish® would be inhalation or dermal exposure during handling. The same exposure pathways would be possible during any unanticipated spills. Mitigation measures in place under Alternatives 2, 3, and 5 that would substantially limit the risk to application workers include:

- A 24 hours/day security effort where the rotenone is stored.
- Enough potassium permanganate (rotenone neutralizer) would be on-hand to neutralize the largest container of rotenone stored on site.
- Certified pesticide applicators would be responsible for all phases of rotenone application.
- The protective equipment listed on the labels of both rotenone formulations will be used by all personnel who handle these products. This includes disposable coveralls, gloves, eye protection, nitrile gloves, and air purifying respirators. Air purifying respirators provide a 10 to 50 fold protection factor. Extra replacements will be available at all times during the implementation phase.
- All of the following detailed plans would be completed according to recommendations and examples provided in the "Rotenone Use in Fisheries Management: Administrative and Technical Guidelines Manual" (Finlayson et al. 2000) prior to project implementation: rotenone application plan, site safety plan, site security plan, and a spill contingency plan.

ENVIRONMENTAL EFFECTS

Direct Effects:

Direct effects to human health are those that occur in the short-term, over a period of several years, and at the immediate site of Diamond Lake itself.

For the general visiting public, no exposure to the fish toxicant is expected, therefore no direct effect to the health of the visiting public is expected under Alternatives 2, 3, and 5. For members of the public who live at Diamond Lake there is a risk of exposure with the possible consumption of contaminated water from the wells on the west side of the lake. Concentrations of rotenone in drinking water are not predicted to be measurable or significant. This is because of the very dilute levels of rotenone that would exist in the lake

(up to 0.10 ppm or mg/L). The worst-case concentration of rotenone to occur in the lake immediately after application is 0.10 mg/L which is below the USEPA PRG[6] safe level of 0.15 mg/L Table 41). The safe level is protective of children and adults drinking 1 and 2 liters of water per day, respectively, for 350 days per year for 30 years. Risks from inert ingredients are difficult to predict, but are likely very low given the large dilution.

Dermal contact of contaminants potentially in the shallow wells while bathing or showering is not predicted to be a significant exposure pathway. Concentrations of rotenone in the lake are projected to be around 0.1 milligram of rotenone per liter of treated lake water (mg/L) which is below the safe level for tap water use established by the EPA (0.15 mg/L) (Table 41). For the chemicals of concern, dermal contact risk would be considered insignificant. Moreover, since rotenone breaks down rapidly in sunlight, and since it is strongly bound to lake sediments, the concentrations that may actually show up in well water is expected to be substantially lower than the concentration in the lake itself. If summer home owners were to drink the dilute concentration over the course of several days, illness is possible but not likely.

The standards used for non-toxic determinations were established by the Environmental Protection Agency. When EPA sets safe levels for tap water use, they build in a margin of safety. The margin of safety establishes a level much lower than any level that has been shown to result in a toxic response to long-term exposure studies of lab animals. The margin of safety is used to protect individuals who might be particularly sensitive or allergic to substances. Sensitive individuals are those that might respond to a lower dose than average, which includes women and children. Human susceptibility to toxic substances can vary by two or three orders of magnitude. Factors affecting individual susceptibility include diet, age, heredity, preexisting diseases and lifestyle.

The margin of safety approach used by the EPA in establishing safe exposure levels takes into account much of the variation in human response. Since EPA has accounted for such variation by choosing very conservative levels of contamination, then no direct adverse effects to women, children, and other sensitive individuals are expected from any alternative that would apply rotenone following label directions.

To further lessen any risks to human health, bottled drinking water would be supplied to the users of all potentially impacted wells if rotenone or any of the associated added ingredients are detected in the monitoring wells on the lake's west shore. Supplied drinking water will only be ceased once it is determined that no detectable levels of rotenone or Noxfish® additives are below detectable levels. If monitoring wells or domestic wells sampling detects any of these chemicals above the USEPA Tap Water PRG, users will be advised of their risks and discontinuance of well use may be enforced. Through temporary closure of Diamond Lake to the visiting public and temporary discontinuance of well use where appropriate, oral exposures would be significantly minimized or eliminated.

[6] PRG - preliminary remedial goal. The level of a chemical in drinking water that is not expected to cause any adverse effects for a lifetime of exposure. Lifetime exposure is based on 30 years of exposure for a child and adult drinking 1 and 2 liters, respectively.

Application workers are most at risk of receiving a dose and becoming sick from Alternatives 2, 3, and 5. If spills on the skin or splashes into eyes, or accidental inhalation occurs and such exposures are not washed off as required or if the victim is not moved to an area of fresh air as required, then application workers could become temporarily ill. In an extreme case of high exposure, death could occur. These potential direct effects to human health are expected to be minimized or avoided all together by following the prudent industrial handling practices required by law and as mitigation measures of this project. Risk to application workers is minimized through ensuring adequate safety measures are taken to minimize exposure. By completing the rotenone application plan, site safety plan, site security plan, and a spill contingency plan, risk to site personnel including ODFW Staff and certified application workers would be substantially reduced to safe levels or eliminated.

Neither Alternatives 1 nor 4 would result in any direct effects to the public or administrative personnel from rotenone or its additives because the fish toxicant would not be applied under either of these alternatives.

Indirect Effects:

The indirect effects associated with Alternatives 2, 3, and 5 involve effects to human health downstream of Diamond Lake and effects to human health over long periods of time.

The water in Diamond Lake is rich in nutrients (nitrogen and phosphorus) largely due to an overabundance of fish. Nutrient rich waters can lead to downstream public health concerns when such waters are consumed. Alternatives 2, 3, and 5 would send nutrient rich waters to downstream areas during the winter draw down period. However, these draw down waters would be delivered during the cooler, wetter winter months of the year when proliferation of plant growth (typically associated with a nutrient flux) is less likely and when the dilution of the lake water would be maximized. Due to the timing of the draw down, no indirect effects to human health associated with downstream water consumption are expected.

Following the draw down and rotenone treatment, the lake is expected to have increased nutrient loads from the decomposition of dead fish and nutrient rich suspended sediments generated by wave action and the other connected activities that would occur when the lake level is 8 feet in elevation lower than normal. Moreover, because zooplankton populations would be killed by rotenone, a short-term increase in phytoplankton abundance is expected along with the water quality problems associated with algae proliferation. Once the lake begins to finally spill into Lake Creek, these short-term interactions may function together to decrease water quality downstream in Lake Creek and Lemolo reservoir. Alternatives 2, 3, and 5 could result in a short-term indirect effect to the health of potential downstream users of Lake Creek with an increased risk of water borne pathogens and associated illness. This short-term potential indirect effect is not expected to be a risk within or downstream of Lemolo reservoir because by the time the nutrient-rich waters reach the reservoir, there would be substantial dilution from small tributaries and from the North Umpqua River that mixes with Lake Creek in Lemolo Lake.

With eliminated or substantially reduced populations of tui chub, Diamond Lake would have lower levels of nutrients, thus lower downstream eutrophication in Lake Creek and Lemolo

Reservoir. Alternatives 2, 3, and 5 have the greatest potential to result in indirect long-term beneficial effects to downstream water quality and the health of the people who may potentially drink from these downstream waters. It is recognized under these alternatives that some chub may remain after the rotenone treatment or may be introduced in the future, such that their populations eventually explode again. The contingency plan is expected to help to slow or alleviate future risks to human health if tui chub do recur.

Neither Alternatives 1 nor 4 would result in any indirect effects to the public or administrative personnel from rotenone or its additives, because the fish toxicant would not be applied under either of these alternatives.

Cumulative Effects from Both Forms of Toxins:

Under Alternative 1, the potential exists for cumulative effects to human health over years of chronic exposure to algal toxins. Uncertainty exists as to accumulation of anatoxin-a in fish tissue, so that fishermen may be at risk in eating their catches (Falconer 1993). Although it does not appear to be the case that recurrent low exposure to anatoxin-a leads to health problems later on, this is not established, and there is concern that people so exposed may become sensitized and develop increasingly more severe reactions with each new exposure (Backer 2002).

Anecdotal evidence for chronic effects of microcystins on humans is based in large part on the high rates of primary liver cancer in the rural regions of China where drinking water was obtained from ditches and ponds with large cyanobacteria loads. Where drinking water supplies have been changed from surface sources to deep wells, cancer rates have begun to drop (although other contributing factors are also being addressed). Other studies have shown microcystins to promote liver tumor development in lab animals. Yet, there is much uncertainty regarding the potential cumulative effects to human health under Alternative 1.

Under Alternatives 2, 3, and 5, the possibility exists that workers exposed to both rotenone and algae toxins, while implementing these alternatives, could become ill or more sensitized to the toxic effects of either of these toxins as a result of exposure to both. This potential cumulative effect would apply to the workers who would be implementing these alternatives and the risk would be over the course of about a month. No studies have been done to confirm this possibility. Over the longer-term, within a few years of implementation, Alternatives 2, 3, and 5 are predicted to result in a beneficial effect to human health, lessening the possibility of chronic cumulative effects of toxic algae blooms compared to Alternative 1. However, if some chub remain after the rotenone treatment or if chub are reintroduced in the future such that their populations eventually explode again, water quality problems and associated health risks would be expected to recur. Implementation of the contingency plan may help to slow or alleviate future risks to human health if tui chub do recur.

Potential cumulative effects to human health are greater under Alternative 4 than Alternatives 2, 3, and 5 because workers are expected to be exposed to toxins over the 7 year lifetime of the project and potentially beyond. Based on recent data from Lava Lakes, (Eilers pers. com) there is considerable uncertainty regarding the long-term effectiveness of

mechanical tui chub removal limiting algae blooms, thus under this alternative there is a higher chance of additive cumulative effects to human health from continued exposure to toxins than under other action alternatives. This is especially true if the same workers were repeatedly exposed year after year to the algal toxins. Additionally, if mechanical removal efforts fail to meet the stated goal of removing 90-95% of the spawning age chub annually or if the removal is stopped, the tui chub population would be expected to quickly rebound. As such, water quality problems and associated health risks would likely recur. Annual implementation of the contingency plan over time would increase the likelihood of achieving or maintaining improved water quality and lowered future health risks.

Cumulative effects are also assessed by evaluating the potential effects of past, present, and reasonably foreseeable actions added to the effects of the various alternatives. In the vicinity of Diamond Lake, other pesticides have been used in the project area in the past. Table 9 shows that the herbicides Cimizine, 2,4-D and Trichlopyr were sprayed along the road shoulders of Highway 138 to clear vegetation between 1980 and 1983. The herbicide picloram was used in the project area when it was spot applied to individual plants or groups of spotted knapweed at several locations using a backpack sprayer along Highway 138 near Diamond Lake and near the south entrance to Diamond Lake. From the mid 1960's to 1982 the pesticide Malathion was applied multiple times each summer to kill mosquitoes at the south shore marsh and various other areas around the lakeshore. Of the above pesticides, only the use of picloram to spray scattered groups of spotted knapweed is reasonably foreseeable in the future in the immediate vicinity of Diamond Lake. The small amount of picloram used in 2003 and expected to be used over the next few years in the vicinity of the project has little chance of entering surface or ground water given the extremely small amounts needed to spray the scattered plant populations and the time of year that spray has been and would continue to be applied (dry summer months, Umpqua National Forest Noxious Weed Environmental Assessment). Moreover, the other pesticides used in previous decades (1960-1980's) have essentially no chance of resulting in any additive cumulative effect to human health over and above any exposure to either rotenone or algae toxins because the previously used pesticides would have disappeared by now.

A multitude of past, present, and reasonably foreseeable projects and activities use various forms of petroleum products that can be harmful to human health. These past and on-going activities and projects include activities such as timber sales, forest fuels reduction projects, forest thinning, hazard tree removal, campground maintenance and improvement, highway construction and reconstructions, paving projects, facility construction and reconstruction, and marina operations and boating (Tables 9, 10 and 11). Any of these that have taken place or are on-going, or that will occur during the implementation of Alternatives 2, 3, or 5, within the watershed of Diamond Lake, could potentially deliver petroleum-type toxicants to surface water and groundwater of the lake. This possibility is heightened if substantial spills were to occur in association with any of the projects. However, mitigation measures built into each of these projects reduces the risk of petroleum spills occurring.

Some of the inert ingredients of the liquid rotenone formulation (trichloroethylene, naphthalene, and xylene) are also present in the fuel of motor boats, and as a result are commonly found in lakes where motorized activities occur. Prior to breakdown of these inert ingredients in Noxfish®, there is a potential of an additive effect from these compounds from

both the Noxfish® and the boat use (during the application and mixing of the powered rotenone that does not contain any inert ingredients). Added together, from both sources, these inert ingredients could potentially reach higher concentrations than if no boats were used in the Lake. However, Finlayson (2000) reported that concentrations of these compounds in water immediately following treatments using Noxfish were low and presented no health risks. As such, the likelihood of additive effects is very low. Alternative 5 poses a slightly higher risk of resulting in an additive effect because it would apply proportionally more Noxfish® than Alternatives 2 and 3. With more use of Noxfish®, there is more chance that people would be exposed to the inert ingredients present in this formulation. If an additive effect did occur it would last a short time over a few weeks, because trichloroethylene, naphthalene, and xylene all break down within about three weeks time (Table 15, in the Lake Ecology Section of this DEIS). Those most at risk of an additive effect would be the application workers involved in implementing Alternatives 2, 3, and 5.

Given long-term exposure to various forms of toxicants in the environment, it is conceivable that human health could be incrementally compromised by long-term exposure to these products in the waters of Diamond Lake. This could potentially result in cumulative effects to human health when added to the effects from toxins potentially received as a result of any of the alternatives associated with this project. However, there is no scientific literature to support this hypothesis.

OVERVIEW AND COMPARISON OF TOXICITY RISKS

The blue-green algae toxins presently found in Diamond Lake and the formulations of rotenone proposed for use in Diamond Lake are both potentially very dangerous to people in terms of acute toxicity. The Environmental Protection Agency has established preliminary remedial goals (PRGs) for rotenone and most of the inert ingredients found in the liquid formulation of rotenone (Table 41) where consumption is not expected to cause any adverse effects for a lifetime of exposure. The USEPA has not established safe levels in water for either of the blue-green algae toxins. There have been fewer investigations into the human health risks of the blue-green algae toxins compared to that for rotenone. In terms of acute toxicity where large doses are received, both rotenone and the blue-green algae toxins can cause serious illness or be deadly to people (Table 43). In terms of chronic, long-term exposure to low levels of these toxins, rotenone has been found to be relatively benign, while not enough data exists for any good conclusions on the long-term effects of exposure to low levels of either anatoxin-a or microcystin.

Table 43. Comparison of Alternatives for Potential Worst-Case Human Health Impacts

Alternatives Toxin type	Alternative 1 algae toxins		Alternatives 2, 3, and 5 algae toxins and rotenone		Alternative 4 algae toxins	
Group	**Adminis-trative workers**	**General public**	**Administrative workers & summer home residents**	**General public**	**Administrative workers**	**General public**
Level and duration of possible exposure	Workers — Exposure to high concentra-tions of algae toxins for many years.	Exposure to low concen-trations of algae toxins, every summer for many years.	Workers — exposure to high concentrations of algae toxin and rotenone for 1 month. Residents — exposure to low concentrations rotenone for 1 month if wells are contaminated and bottled water not used.	No exposure to either toxin due to draw down and strict controls	Workers — exposure to high concentrations of algae toxins for at least 7 years with incremental declines thereafter if mechanical chub removal is effective and it continues.	Exposure to low concen-trations of algae toxins, every summer for at least 7 years with incremental declines thereafter if mechanical chub removal is effective and it continues.
Potential con-sequences if exposed	Workers — serious illness or death	Mild to serious illness; latent long-term effects unknown.	Workers — serious illness or death Residents — potential illness	None	Workers — serious illness or death Residents — potential illness; latent long-term effects unknown	Mild to serious illness, latent long-term effects unknown